

# *Tetracapsuloides bryosalmonae* (PKD)

## I. Causative Agent and Disease

Proliferative kidney disease (PKD) is caused by the PKX cnidarian myxozoan (Malacosporea), *Tetracapsuloides bryosalmonae*, that is a parasite of freshwater bryozoans (*Fredericella* sp., *Plumatella* sp.) and salmonid fish. Waterborne spores of *T. bryosalmonae* are released from the bryozoan host where they infect the fish host, primarily through the gills. The parasite travels via the blood to the kidney and other vascular organs where it proliferates, causing chronic inflammation often accompanied by secondary pathogen infections.

## II. Host Species

PKD has been reported in both wild and captive salmonids and several other species including whitefish and northern pike in the Pacific Northwest and Newfoundland; trout, Atlantic salmon and grayling in Europe, including Finland and Sweden; Arctic char in Iceland. In Alaska, the parasite has been reported in lake-reared juvenile sockeye salmon and two adult returning chum salmon.

## III. Clinical Signs

The gross clinical signs of PKD include pale gills, a uniformly swollen kidney (may be gray/mottled) and spleen with exophthalmia, ascites and anemia.

## IV. Transmission

Spores, released from freshwater bryozoans, infect salmonids through the skin and gills releasing ameoboid sporoplasms. A single spore is sufficient to infect a fish and cause clinical PKD. These travel to the kidney and undergo extra-sporogonic multiplication in the interstitium and differentiate through sporogenesis in the kidney tubules. Resulting spores, designated fish mala-

cospores, are released with the urine to infect more bryozoa. Vertical transmission allows *T. bryosalmonae* to persist in the bryozoan host. Brown trout are known to be subclinical carriers for at least 5 yrs.

## V. Diagnosis

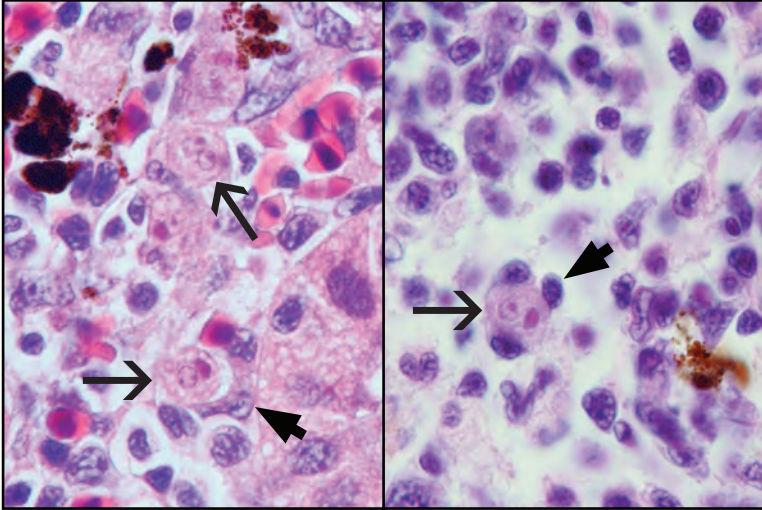
Microscopic diagnosis is made by: Giemsa-stained imprints showing amoeboid PKX cells (10-20 um) with foamy cytoplasm, distinctive cell membrane and 1 mother cell (primary) nucleus with 1-7 daughter cells; histological exam indicating proliferative and granulomatous nephritis, vascular necrosis and thrombi with eosinophilic PKX cells among the kidney interstitial cells, often surrounded by attached host macrophages. Parasite DNA can be detected in all organs by PCR and PKX cells can be observed in kidney, spleen and liver of infected fish by immunohistochemistry.

## VI. Prognosis for Host

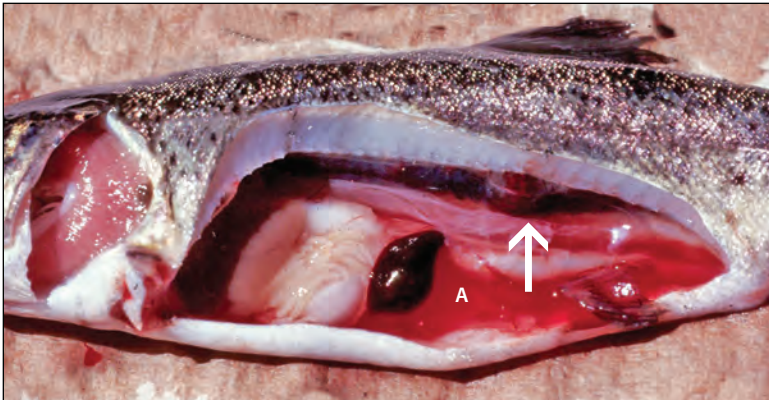
Temperature increase induces transition from covert into overt infection where infectious stages of *T. bryosalmonae* develop and are released into the water. Variable mortality (5-90%) occurs at elevated water temperatures (12-15°C) while fish that show less severe clinical signs of disease largely survive the infection when water temperature is lower (< 12°C). Surviving fish develop immunity and may clear the infection with regeneration of damaged tissues. The decline of wild salmonid populations in several rivers has been attributed to PKD which will become an emerging fish pathogen as global warming continues.

**VII. Human Health Significance**

This parasite is a pathogen for fish.  
There are no human health concerns associated with PKD.



Histology of eosinophilic PKX cells (arrows) in the kidney interstitium of steelhead trout containing large primary cell nuclei (dense red) next to translucent daughter cell nuclei. PKX cells are surrounded by basophilic (blue) host inflammatory cells (arrowheads); H&E, X 1000.



PKD in rainbow trout exhibiting bloody ascites (A) and swollen nodular posterior kidney (arrow) due to inflammation and proliferation of PKX cells (photo: M. L. Kent and R. P. Hedrick, Univ. of Calif., Davis).